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Water Flux From Partial-Thickness Skin Wounds: Comparative Study of the Effects of Er:YAG and Ho:YAG Lasers

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Background and Objective: **The clinical use of lasers to cut and coagulate tissue necessitates a better understanding of how the residual thermal damage affects healing. This study was designed to evaluate the effects of varying degrees of thermal damage on the healing process.**
Study Design, Materials and Methods: **Partial-thickness lesions were created in guinea pig skin using an Er:YAG laser, a Ho:YAG laser, and a scalpel. To monitor recovery of the stratum corneum, water flux from the wound sites was quantified and histological data obtained for approximately one week.**
Results: **The data indicate an exponential water loss pattern from all wounds. Water flux from the scalpel- and the Er:YAG laser-induced wounds was initially high but decreased rapidly with decay rates (mean ± SE) of $0.46 \pm 0.01\ day^{-1}$ and $0.38 \pm 0.01\ day^{-1}$, respectively. The Ho:YAG laser-induced wounds demonstrated a different pattern of decay with lower water flux values initially and a decay rate of only $0.13 \pm 0.01\ day^{-1}$.**
Conclusion: **Histological and water flux data reveal that Er:YAG laser-induced wounds achieve epidermal integrity only slightly after scalpel-induced lesions, and Ho:YAG laser-induced wounds heal substantially slower and contain more granulation tissue.**

Key words: evaporimeter, insensible perspiration, transepidermal water loss, thermal damage

INTRODUCTION

The presence of eschar or necrotic tissue in a wound bed delays healing as epidermal cells must migrate deep into the tissue to reach a viable bed. The amount of thermal damage present in laser-induced skin lesions thus affects the rate of repair. Previously, it was shown that the 20–50-μm-wide thermal damage zone produced by an Er:YAG laser did not significantly alter healing compared with scalpel-induced lesions [1,2]. Thermal damage zones larger than ~300 μm, such as those produced by continuous-wave CO_2 lasers, have been associated with an extensive inflammatory response, delayed healing, and increased scarring [1,3–6]. An intermediate amount of thermal damage may inhibit tissue desiccation by limiting the amount of water lost through injured skin and thus avoid the problems associated with dry wound healing [7–9] without impeding epidermal migration. To further understand the relationship between the width of the coagulated zone and skin wound healing, we followed the wound healing process histologically and monitored recovery of the stratum corneum by measuring water flux rates from the wound site.

The stratum corneum performs a variety of protective functions including the prevention of excess water loss through skin [10]. As a result of its water barrier function, damage to this outermost layer of the skin affects its ability to limit water loss from the underlying tissues to the ex-

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ternal environment. The resulting dehydration produces necrotic tissue that delays healing because the epidermal cells must migrate under the debris to find moist, living cells across which they can advance before reepithelialization can occur. Problems associated with uncontrolled water loss from wound sites also include increased scarring and excessive heat loss caused by the evaporation of water and resulting in higher metabolic demands [11,12].

Transepidermal water loss is defined as "the rate at which water migrates from the viable dermal tissues through the layers of the stratum corneum to the external environment" [13]. The measurement of transepidermal water loss is considered a useful means of assessing the repair process of the epidermal water barrier [13–19]. Repair can be followed because the formation and maturation of a new stratum corneum is characterized by a reduction in water loss rates from their elevated postinjury values. A return to baseline levels indicates that the skin has healed and regained its protective water barrier function.

Numerous techniques for measuring transepidermal water loss have been employed [13, 20,21]. The early ventilated and unventilated chamber techniques interfered with the microclimate near the skin surface and therefore affected the very parameter being measured. The development of an open chamber device in the mid-1970s and now available commercially as the ServoMed Evaporimeter helped alleviate this problem [20]. The evaporimeter calculates water flux rates by determining the water vapor pressure gradient adjacent to the skin's surface. When measured within the boundary layer of stagnant air above the skin, the gradient is proportional to the amount of water evaporating from the surface. To obtain accurate and reproducible water flux data, careful control of individual and environmental factors such as body site, skin temperature, air flow, ambient temperature, and relative humidity must be maintained [11,22–25]. Previous studies have generally failed to demonstrate sufficient regulation of environmental conditions to provide reliable data. In addition, problems with underestimation have been described when using an evaporimeter to measure rates greater than ~75 g m^{-2} h^{-1} [26,27]. Furthermore, since evaporimeters calculate a water flux rate based on the total area of the probe's measuring chamber, actual wound size is an important but frequently overlooked consideration. Normalizing the water flux rate to account for the percentage of injured tissue covered by the probe measuring chamber has not previously been documented.

Several studies using ventilated and unventilated chamber techniques to evaluate water flux rates from injured skin have described a two-phase pattern of water barrier regeneration: one phase characterized by a rapid decrease in water loss from elevated postinjury levels followed by a second phase distinguished by a markedly slower change in water loss rates as values gradually decreased to normal [16–19]. A recent study using an evaporimeter to monitor wound healing in miniature pigs also suggested a biphasic pattern of repair with the point on the water loss versus time curve where the steep slope of the first phase changed to the more gentle slope of the second phase correlating with histological reepithelialization [28]. Water flux rates measured from the 3-mm punch-biopsy wounds never exceeded 65 g m^{-2} h^{-1} and problems with evaporimeter underestimation were thus avoided. One study comparing the effect of two different wound coverings on guinea pigs described a triphasic water loss pattern [29]. The first phase was characterized by constant elevated water loss rates that were attributed to a delay in establishing primary epidermal closure. The second and third phases corresponded to the biphasic pattern discussed previously. Using an evaporimeter, initial water flux rates exceeding 100 g m^{-2} h^{-1} from 2 cm × 2 cm wounds were reported. Such high values would indicate that the actual water flux rates were underestimated; this artificial suppression may have resulted in the constant "phase one" period observed.

To evaluate the effects of varying degrees of laser-induced thermal damage on healing, the water flux through Ho:YAG-, Er:YAG-, and scalpel-induced lesions was monitored and correlated with a histological study of wound repair. The partial-thickness wounds were created in vivo in guinea pig skin and data were collected for ~1 week. The relationship between water loss and the histologically observed healing process was assessed.

MATERIALS AND METHODS

The backs and flanks of eight 500–600 g female guinea pigs (Charles River Breeding Laboratories, Hartley strain) were shaven with electric clippers and chemically epilated (Nair) no more than 24 hours before surgery. Immediately prior to surgery, each animal was anesthetized with Xylazine (2 mg/kg), Ketamine (35 mg/kg), and Atro-

pine (0.04 mg/kg) administered intramuscularly. Eight to ten 4 mm × 4 mm middermal lesions placed ~2 cm apart were then created forming two rows parallel to the spine. One row of wounds on each guinea pig was made with a #10 stainless steel scalpel blade using a freehand surgical technique. On four of the animals, the second row of wounds was created using 2.94-μm radiation from an Er:YAG laser (Schwartz Electro-Optics, Laser 1-2-3). On the remaining four animals, the second row of wounds was created with 2.10-μm radiation from a Ho:YAG laser (Schwartz Electro-Optics, Laser 1-2-3). Each laser emitted ~250-μs-long pulses at a pulse repetition rate of 2 Hz. For both lasers the radiation was focused with a 125-mm-focal-length, biconvex, BaF_2 lens to achieve a 1-mm-diameter spot as measured by examining the ablation crater produced in guinea pig skin by a single pulse. A computer-controlled, stepper-motor-driven, X-Y translator (Klinger Scientific, CD 4) was used to create the 4 mm × 4 mm laser-induced lesions. A program was written to ensure the maximum amount of overlap between adjacent spots and to create a uniform wound depth. Reproducibility of laser- and scalpel-induced wounds was confirmed by histological analysis of the biopsied lesions. In all cases, postoperative hemostasis occurred naturally within ~1 minute and the wound sites were left uncovered throughout the experiment.

The optimum parameters for creating partial-thickness lesions in guinea pig skin with both lasers were determined from preliminary studies. The Er:YAG laser-induced wounds were created by delivering six pulses per site with a fluence per pulse of 20 J cm^{-2} via an articulated arm that was scanned across the guinea pig stepwise in 408-μm increments at a rate of two steps per second. The Ho:YAG laser-induced wounds were created by delivering three pulses per site with a fluence per pulse of ~60 J cm^{-2} via two mirrors that reflected the beam through the focusing lens. Here, the beam was fixed and the guinea pig was positioned on the translator that moved in horizontal and vertical steps of 577 μm at a rate of two steps per second. In both cases, fluence was calculated from the pulse energy detected with a joulemeter (Gentec, ED-200) and the measured spot size.

Several lesions were created with each modality so that the correlation between water loss rates and histologically observed healing could be assessed. Biopsies were taken of all three wound types on days 0, 1, 3, 5, and 7, as well as on days 9, 11, and 14 in the case of the Ho:YAG laser-induced wounds. The 6-mm diameter punch biopsies were taken under local anesthesia (intradermal 1% Lidocaine) and immediately fixed in 10% formalin. The biopsy sites were closed with one or two stitches using 4-0 nylon suture on an FS-2 cutting needle. The biopsy specimens were processed in graded alcohols and xylenes, mounted in paraffin blocks, sectioned to display the lesions in cross section, and stained with hematoxylin and eosin. The specimens were then viewed under light microscopy, and the loss of birefringence in the denatured collagen fibers was used to determine the width of the thermal damage zone.

An evaporimeter (ServoMed, Ep1D) was used to quantify water flux following published guidelines [24]. All measurements were taken in an anechoic chamber; room temperature and relative humidity were monitored with a digital thermometer (Omega, HH81) and evaporimeter, respectively. Data were recorded from the laser- and scalpel-induced lesions designated as the final biopsy sites. During water loss measurements, each guinea pig was confined in a fitted Plexiglas box designed to expose the wound sites. The animals were not anesthetized but only constrained by the walls of the Plexiglas box. The gold-plated protection cover with screen and grid attached (ServoMed, #2107) was placed on the evaporimeter probe. The probe itself was mounted on a burette clamp, attached to an X-Y-Z translator (Newport, 460-XYZ), and positioned to maintain a constant light pressure against the skin. The probe, guinea pig, and Plexiglas box were all placed inside a large clear-plastic box with an open top that served as a draught shield. The evaporimeter was connected to a strip chart recorder (Gould, 2200S) to provide an analog output of the water loss rates while values were recorded manually from the evaporimeter's digital display. As recommended in published guidelines [24], data were recorded when the initial fluctuations in TEWL values stabilized without the use of the built-in filtering elements. Such stabilization was typically evident ~60–90 seconds after the probe was placed against the skin, although times were somewhat longer during the early readings when evaporation rates were high. The value displayed during the 30-second period following stabilization was recorded as the actual water flux rate.

To avoid the hypotensive and hypothermic effects of the anesthesia, water flux measurements did not begin until the animals were ambulatory (approximately 3 hours postoperatively)

and in no case were they taken less than one hour after a biopsy. Measurements were initially taken at 4-hour intervals, and as the rate of change of water flux rates decreased the measurement interval increased to 6, 12, and finally 24 hours. At every measurement session three water flux rates were recorded: one each from the laser- and scalpel-induced wounds and one from normal skin. The laser and scalpel measurement sites were photographed every 24–48 hours. Wound area was quantified from the photographs using a digitizing tablet (Numonics, 2210-0.30.C) and data reduction software (Jandel Scientific, Sigma-Scan v.3.90).

Throughout the experiment each guinea pig was housed separately and given free access to standard guinea pig chow and water. The animals tolerated the initial surgery and the measurement procedures well with no evidence of infection. Data collection continued until the wound crusts began to detach and water flux rates approached baseline values. When data collection was completed, the animals were euthanized with an overdose of Pentobarbital (≥150 mg/kg).

RESULTS

Stable environmental conditions were maintained in the anechoic measuring chamber throughout data collection. During a single measurement period the maximum temperature and relative humidity variations were 1.8°C and 10%, respectively, with mean differences of only 0.8°C and 3%. The areas of the Er:YAG laser-, Ho:YAG laser-, and scalpel-induced lesions used for water flux measurements were 0.18±0.02 cm^2 (mean ± SD, n = 4), 0.12±0.01 cm^2 (n = 4), and 0.16±0.03 cm^2 (n = 8) initially and decreased to 0.15±0.03 cm^2, 0.11±0.01 cm^2, and 0.13±0.02 cm^2 by day 7.

All water flux rates recorded by the evaporimeter were normalized to compensate for intra- and interindividual variations in baseline values due to metabolic or environmental changes. The evaporimeter calculates water loss rates from the flux through the entire area of the probe's measuring chamber. Since the lesions were made smaller than the ~1 cm^2 chamber area to avoid exceeding the 75 g m^{-2} h^{-1} accuracy limit of the device, variations due solely to differences in wound size were removed by determining water flux rates from the actual wound area only. To accomplish the normalization, the difference between the actual water flux rate recorded from each wound site and from normal skin was divided by the ratio of the wound area to the probe's measuring chamber area. All rates presented herein thus represent a deviation from baseline, and a reported rate of 0 g m^{-2} h^{-1} indicates a return to normal water loss and normal stratum corneum water barrier function. The water flux rate measured from the uninjured guinea pig skin of all eight animals at every measurement session was 3.6±1.2 g m^{-2} h^{-1} (mean ± SD, n = 216).

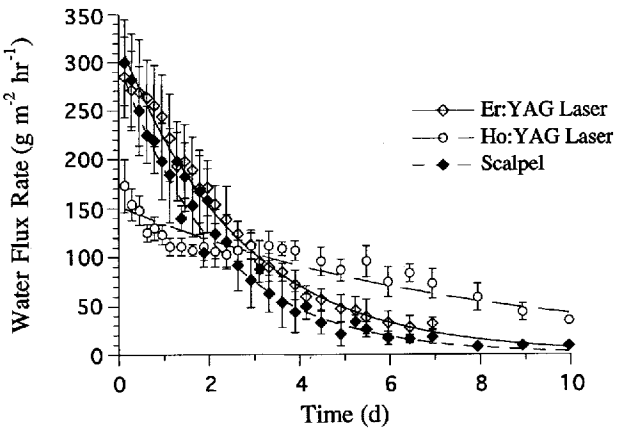

Fig. 1. Water flux rates normalized to that measured from normal guinea pig skin. Solid and dashed lines represent a least-squares exponential fit to the data. Values reported as mean ± SD.

The water flux data collected from laser- and scalpel-induced skin lesions are presented in Figure 1. Although the patterns from the scalpel- and the Er:YAG laser-induced wounds are similar, the pattern from the Ho:YAG laser-induced wounds is clearly different with a flux rate that was lower initially and decreased more slowly over time. The Ho:YAG laser-induced lesions had an initial water flux rate of 173±27 g m^{-2} h^{-1} (mean ± SD, n = 4), differing markedly from the 285±42 g m^{-2} h^{-1} (n = 4) and the 300±45 g m^{-2} h^{-1} (n = 8) from the Er:YAG laser- and scalpel-induced wounds, respectively. The somewhat large standard deviations were expected due to the individual nature of transepidermal water loss as documented by Pinnagoda et al. [24] and Blichmann and Serup [22] who concluded that interindividual variations could be large and therefore only relative data should be considered when comparing water flux rates from different subjects.

Although actual water flux rates differed somewhat from animal to animal, the general trends are consistent. In each case the data demonstrate an exponential decay of the form:

$$\text{water flux} = ke^{-at}$$

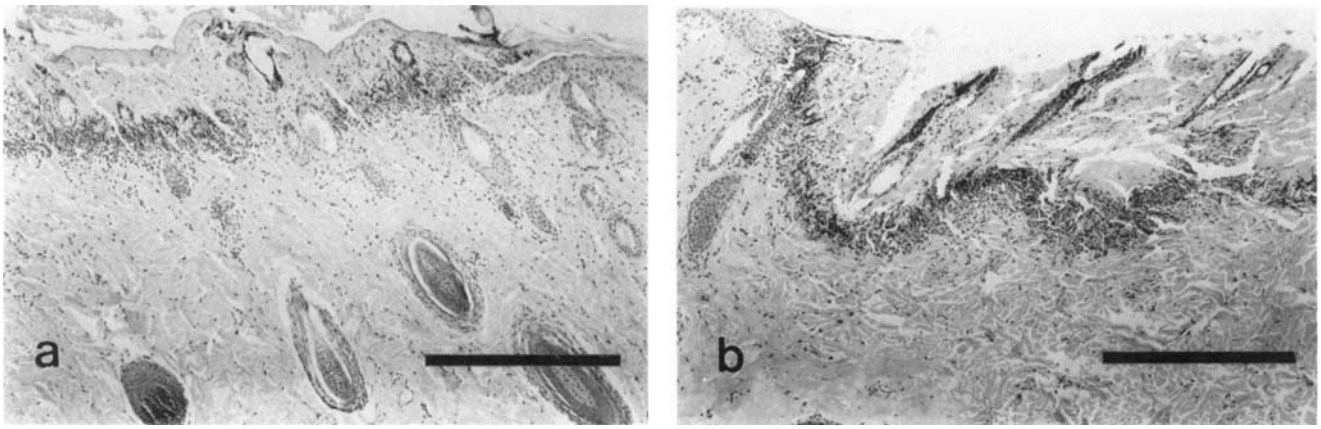

Fig. 2. Photomicrograph of (a) Er:YAG laser- and (b) scalpel-induced wounds 24 hours postoperatively. Note margination of PMNLs to the base of the wound (hematoxylin and eosin stain, bars = 500 μm).

where k is an individual constant and a is 0.13 ± 0.01 day^{-1} (mean ± SE, n = 105) for the Ho:YAG laser-, 0.38 ± 0.01 day^{-1} (n = 111) for the Er:YAG laser-, and 0.46 ± 0.01 day^{-1} (n = 201) for the scalpel-induced lesions. The decay rate from the Ho:YAG laser-induced lesions indicates a dramatically slower decline of water flux rates over time compared to the Er:YAG laser- and scalpel-induced wounds. The difference between all three decay rates is statistically significant ($P < 0.001$) [30]. As confirmed by the small standard errors, the rate of decay of water flux rates is consistent between animals for a given modality.

Histological data from the middermal, laser-induced lesions indicated a zone of thermal damage ~50 μm wide for the Er:YAG laser and ~650 μm wide for the Ho:YAG laser. By day 1, the inflammatory response was evident in both Er:YAG laser- and scalpel-induced wounds with the infiltration of polymorphonuclear leukocytes (PMNLs) and a 1–2 mm erythematous zone around the wound sites. The PMNLs in the lesions created with the Er:YAG laser migrated to within 50–150 μm of the thermally damaged tissue, but the presence of birefringent collagen between the PMNLs and the thermally damaged tissue indicated that tissue beyond the original zone of thermal damage would be sloughed (Fig. 2a). Whereas the PMNL layer in the Er:YAG laser-induced wounds was ~100 μm wide, histology from the scalpel-induced lesions showed an ~50-μm-wide layer of PMNLs demarcating ~300 μm of tissue to be sloughed (Fig. 2b).

Virtually no cellular response was observed at any of the Ho:YAG laser-induced wound sites until day 3. At this time, the inflammatory response was still minimal with a 50–100-μm-wide PMNL layer beginning to demarcate the region to be sloughed (Fig. 3). Once again, the presence of birefringent collagen in the 150–250-μm zone between the PMNLs and the thermally damaged tissue confirmed the loss of tissue beyond the original thermal damage zone.

A hyperplastic neoepidermis was present on day 3 in the Er:YAG laser- and scalpel-induced wounds, although it was incomplete and apparently poorly attached to the underlying tissue. By day 5, reepithelialization was essentially completed in all Er:YAG laser- and scalpel-induced wounds and local erythema was virtually nonexistent. Whereas the epidermis was still apparently poorly attached in the Er:YAG laser-induced wounds, those created with a scalpel appeared to show good epidermal attachment as well as the presence of an intact stratum corneum and ~150 μm of granulation tissue. Day 7 histology showed a well-attached stratum corneum and 400–500 μm of granulation tissue at the Er:YAG laser-induced wound sites. The epidermis at both the Er:YAG laser- and the scalpel-induced wound sites was still hyperplastic, measuring 100–200 μm across compared to a normal thickness of ~25 μm.

Day 7 histology from the Ho:YAG laser-induced wounds showed a diffuse inflammatory response, incomplete reepithelialization, and a hyperplastic epidermis. Reepithelialization and granulation tissue formation continued until day 11 when a hyperplastic but well-attached epidermis complete with stratum corneum layer was observed. Histological sections from day 14 in one

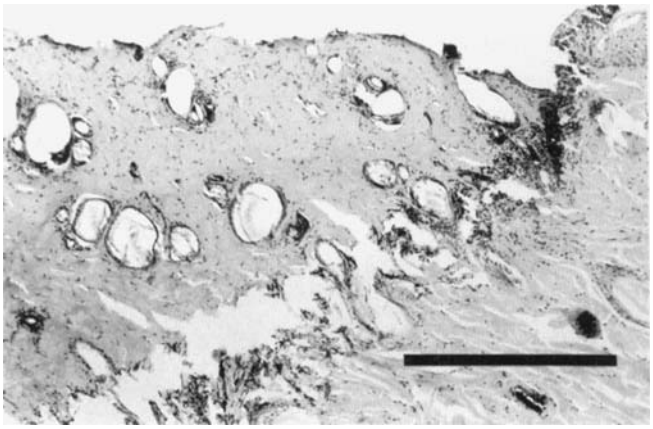

Fig. 3. Photomicrograph of Ho:YAG laser-induced wound 3 days postoperatively. Note margination of PMNLs to the base of the wound (hematoxylin and eosin stain, bar = 500 μm).

animal showed granulation tissue extending to the full thickness of the skin, in many regions to a depth of ~1,200 μm.

Histological data from both laser- and scalpel-induced lesions confirmed that the base of the acute damage zone was in the middermal region. In addition, epidermal migration clearly initiated from the wound edges in all three types of lesions. In no case was migration found to initiate from a follicular source.

Gross observation of the wound sites 3 hours postoperatively indicated a region of swelling 1–2 mm wide surrounding the Ho:YAG laser-induced lesions, which were also covered with a thick crust that occasionally demonstrated the presence of char. Redness and swelling during the early inflammatory response were slightly more pronounced around the Er:YAG laser-induced wounds than around the scalpel-induced wounds in several of the animals, but this situation often equalized or reversed itself during healing. Overall, redness and swelling were more pronounced and continued longer around wounds created with the Ho:YAG laser than around those created with a scalpel or the Er:YAG laser. In addition, the Ho:YAG laser-induced wounds were surrounded by a bluish-white region ~1 mm wide that was in turn surrounded by a 1–2 mm erythematous zone. It appears that the bluish-white region corresponded to thermally damaged tissue present at the wound sites.

Finally, it should be noted that the crusts at the scalpel- and Er:YAG laser-induced wound sites typically began to recede around the edges on days 5 and 6, although some were still fully intact on day 7. These variations did not appear to be related to the modality used to create the wound but rather to the individual guinea pig since both crusts on a single animal receded at approximately the same time. The Ho:YAG laser-induced wound crusts, however, generally remained fully intact until at least day 6 or 7. Since the water flux rates toward the end of the healing process revealed little more than a very gradual return to baseline, the wounds were only monitored until the crusts detached or significantly receded. The fact that water flux rates had not completely returned to baseline by the end of data collection indicated that the stratum corneum had not fully regained its water barrier function.

DISCUSSION

The water flux data combined with the histological findings demonstrate that although the 50-μm-wide thermal damage zone produced by the Er:YAG laser did not dramatically interfere with wound healing, it also did not provide any additional barrier to water loss when compared to scalpel-induced controls. The histological results confirm previous findings that Er:YAG laser- and scalpel-induced lesions follow approximately the same time course for healing with reepithelialization completed by day 7 [1]. The presence of birefringent collagen in the region demarcated by the PMNLs in the Er:YAG laser-induced wounds demonstrated the loss of collagen that was not thermally damaged. In fact, three to four times more tissue was sloughed than was thermally damaged by the Er:YAG laser. It should be noted that the denaturation of collagen occurs at ~65°C, whereas cytotoxicity for slow thermal heating begins at 45°C. One could thus argue that the extent of thermal injury cannot be determined by birefringence since cellular damage would be expected to extend beyond the zone of denatured collagen. However, the heating time involved in creating the laser-induced wounds under investigation is very short and the damage due to slow thermal heating may not be significant. It is clear that the effect of thermal cytotoxicity when the heating time is short, such as during pulsed laser ablation of tissue, is a subject requiring additional study.

Another explanation for the sloughing of tissue beyond the zone of denatured collagen in laser-induced wounds is desiccation. Such an explanation is supported by the fact that wounds created with a scalpel also suffered a significant amount of tissue loss (≈300 μm). Furthermore, water flux

rates initially recorded from Er:YAG laser- and scalpel-induced wound sites were substantially elevated above normal rates. Thus, although the Er:YAG laser left a viable wound bed, the laser-induced coagulum was not able to limit water loss. Consequently, there was dehydration of the viable tissue and a subsequent increase in tissue loss. Similarly, two infiltration zones have been reported in experimental burn injuries: a primary zone associated with the burn itself and a second, deeper demarcating zone believed to be due to desiccation of the denuded dermis [31].

The 600–700-μm-wide zone of thermal damage produced by the Ho:YAG laser was associated with lower initial water flux rates from the wound sites and a much slower decay with time. The amount of damage was so great, however, that the healing process was significantly delayed as epidermal cells were forced to travel a greater distance to migrate beneath the debris. In addition to delayed reepithelialization, the cellular inflammatory response in the Ho:YAG laser-induced wounds was delayed relative to the response observed at the Er:YAG laser and scalpel-induced wound sites. Thus, although the thermal damage produced by the Ho:YAG laser did provide an additional resistance to water loss, the wound bed was filled with so much debris that repair was delayed and again significant tissue loss occurred.

The initial water flux rates from wounds created with the Ho:YAG laser were only ~60% of the rates measured from wounds created with either the Er:YAG laser or a scalpel. In fact, water loss from the scalpel- and Er:YAG laser-induced wounds exceeded that from the Ho:YAG laser-induced wounds until day 2 or 3, after which the Ho:YAG laser rates were consistently higher. Whereas water flux rates from the Ho:YAG laser-induced lesions declined slowly over time, rates from the scalpel- and Er:YAG laser-induced wounds rapidly approached the baseline rates measured from uninjured skin. In all cases water flux rates were found to decrease exponentially. The time constant of decay or $(\text{decay rate})^{-1}$ can be used as a measure of the healing time and is 2.6 days for the Er:YAG laser- and 2.2 days for the scalpel-induced wounds. The slower return of stratum corneum integrity in the Er:YAG laser-induced wounds as indicated by the longer time constant was confirmed histologically. By day 5 postoperatively, the scalpel-induced lesions appeared to show good epidermal attachment with an intact stratum corneum, whereas the new epidermis on the Er:YAG laser-induced wounds was still apparently poorly attached. The data from the Ho:YAG laser-induced wounds indicate a substantially slower repair process with a time constant of 7.7 days and histologically observed reepithelialization continuing until day 11. Although the difference observed when comparing histological data and water flux rates from Er:YAG laser- and scalpel-induced wounds is not visually striking, there can be no doubt that the difference between the water flux decay rates is statistically significant ($P < 0.001$). Careful measurement of water flux rates can apparently lead to differentiation of similar healing processes.

In attempting to correlate water flux rates with histologically observed healing for varying degrees of thermal damage, the water flux data collected from both laser- and scalpel-induced wounds failed to confirm the expected biphasic pattern described by other researchers. The scalpel and Er:YAG laser data strongly adhered to a pattern of exponential decay ($R = 0.95$) and had similar decay rates. The Ho:YAG laser data could also be fit to an exponential curve, although perhaps not quite as well ($R = 0.88$), and the decay over time was much more gradual. Although the healing process is typically described as occurring in three overlapping phases, namely inflammation, repair, and matrix formation/remodeling, the return of stratum corneum integrity as determined by the restoration of its water barrier function occurs continuously over time at a rate determined by the nature of the original tissue insult and the amount of debris (or thermal damage) in the wound bed. As the amount of debris or damage increases, the speed of epidermal migration decreases. The resulting delay in healing slows the return of normal barrier function and is characterized by a slower return to baseline water flux rates.

Previous reports concerning quantification of water loss through the coagulum produced by laser irradiation of tissue could not be found. Water flux data from burns and donor sites, however, have been recorded using a ventilated chamber device [32]. Figure 4 illustrates the water flux rates obtained from superficial second degree burns, third degree burns, and donor sites. In the graph, the reported values of water loss from normal skin were subtracted from the actual readings so that all data represent a deviation from baseline. As confirmed by the rather high baseline values (reported as 13 ± 2 g m^{-2} h^{-1}), the use of a carrier gas in ventilated chamber devices produces elevated evaporation rates from skin. Nev-

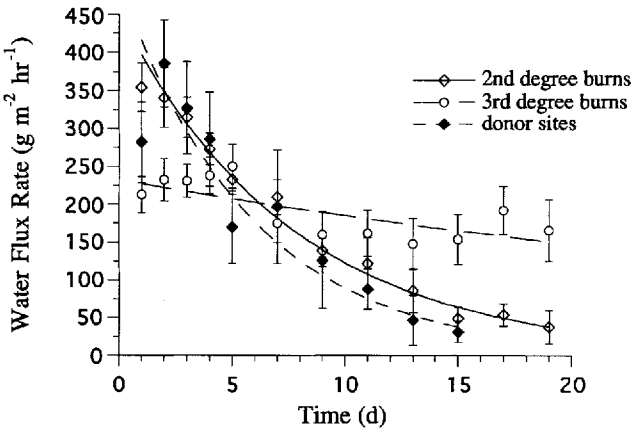

Fig. 4. Water flux rates from superficial second degree burns, third degree burns, and donor sites. Solid and dashed lines represent a least-squares exponential fit to the data. Values reported as mean ± SE; adapted from Lamke and Liljedahl, 1971 [32].

ertheless, other than a longer time frame due to the larger area of injured skin, the burn and donor site data resemble that shown in Figure 1. In particular, water loss from donor sites was reported as being equivalent to that measured from superficial second degree burns just as the water flux rates from scalpel- and Er:YAG laser-induced lesions were found to be very similar. Furthermore, the initial water flux rate from third degree burns was ~75% of that from donor sites compared to the 60% difference between Ho:YAG laser- and scalpel-induced lesions. Thus the similarity in water flux patterns between second and third degree burns and Er:YAG and Ho:YAG laser-induced lesions and donor sites and scalpel-induced lesions indicates a similar pattern of tissue insult and repair.

A key factor in establishing the appropriate environment for tissue repair is the moisture balance at the wound surface. Even though excess water loss causes tissue desiccation and delays healing, some evaporative water loss is normal and a completely impermeable wound covering or dressing causes fluid to accumulate at the wound site. Such excess moisture can loosen a dressing, cause maceration of the surrounding skin, and increase the risk of infection [12,33]. Therefore, an ideal wound covering should allow sufficient water flux to support the growth of new tissue and prevent unwanted fluid retention but not so much that dehydration of the wound bed and therefore excess tissue desiccation occurs. A variety of externally applied dressings do exist, but the thermal damage produced when lasers are used to cut or ablate tissue may serve as a natural dressing if it provides the appropriate barrier to water loss. Obtaining the appropriate moisture balance may then be achieved by selecting wavelength and irradiation parameters that provide the needed degree of thermal damage.

It had been hypothesized that the thin layer of thermal damage produced by a normal-spiking-mode Er:YAG laser inhibited desiccation of viable tissue and thus served as a natural wound dressing [1]. Contrary to the prior results, however, an extension of the initial damage zone was observed from day 0 to day 1 for both the Er:YAG laser- and the scalpel-induced lesions described here. The 600–700-μm-wide thermal damage zone produced by the Ho:YAG laser did result in lower water loss rates initially, but it also delayed the repair process. It is therefore suggested that a laser creating an intermediate amount of thermal damage is needed to precisely cut or remove tissue while leaving a residual zone of thermal damage that prevents excess water loss without delaying healing. In addition to the hemostatic properties provided by lasers, benefits of a natural wound dressing over externally applied bandages include the elimination of the need for frequent dressing changes that may further damage a fragile new epidermis as it reforms and practicality in cases where it is inconvenient or impossible to maintain external bandages such as when the area to be covered is quite large. A laser that may provide the desired degree of thermal damage is the pulsed CO_2 laser. Preliminary results indicate that it provides rapid, precise, bloodless excision of normal and burned skin, and that its 85-μm-wide zone of residual thermal damage does not interfere with wound healing or skin graft take [34,35]. Potential applications of such a laser include skin deepithelialization, microscopic skin cancer surgery, and burn debridement for the preparation of viable skin graft beds.

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